A Prospective Study of

FUNCTIONAL OUTCOME OF SERIAL CAST CORRECTION OF CONGENITAL TALIPES EQUINOVARUS BY PONSETI METHOD

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI – 600 032

In partial fulfillment of the regulations for the award of the

M.S. DEGREE BRANCH - II

ORTHOPAEDIC SURGERY

GOVERNMENT MOHAN KUMARAMANGALAM MEDICAL COLLEGE, SALEM

APRIL 2015

CERTIFICATE

This is to certify that **Dr. A.GURUPRASATH**, Postgraduate student (2013-2015) in the department of Orthopaedics, Government Mohan Kumaramangalam Medical College, Salem has done this dissertation "***A Prospective Study of*** **FUNCTIONAL OUTCOME OF SERIAL CAST CORRECTION OF CONGENITAL TALIPES EQUINOVARUS BY PONSETI METHOD"** under my supervision in partial fulfillment of the regulation laid down by the Tamilnadu Dr. M.G.R Medical University, Chennai, forM.S.,(Orthopaedics) degree examination to be held during April 2015.

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DECLARATION

I, **Dr. A.GURUPRASATH**, solemnly declare that this dissertation titled "*A Prospective Study of* **FUNCTIONAL OUTCOME OF SERIAL CAST CORRECTION OF CONGENITAL TALIPES EQUINOVARUS BY PONSETI METHOD"** is a bonafide work done by me, at Government Mohan Kumaramangalam Medical College, Salem between the period 2013-2015, under the guidance of my unit Chief **Prof. Dr.C.KAMALANATHAN M.S.(Ortho)**, professor of Orthopaedic Surgery. This dissertation is submitted to Tamilnadu Dr. M.G.R Medical University, towards partial fulfillment of regulation for the award of M.S.Degree (Branch – II) in Orthopaedic Surgery.

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MINUTES OF THE MEETING.

Ethical Committee Meeting held on 06.12.2013 at 11.00 A.M.in the Dean's Chamber, Government Mohan Kumaramangalam Medical College, Salem 30.

The following Members were attended.

CHAIRMAN

Dr.A.Karthikeyan, MD., - Dean.

MEMBERS.

1. Dr.S.Mohammed Musthafa .M.D., - Vice Principal.
2. Dr.T.Swaminathan, M.S., - Medical Superintendent.
3. Dr.Priya Jayapal, M.D., HOD of Biochemistry.
4. Dr.R.Jayalalitha, M.D., HOD of Pharmacology.
5. Dr.M.Poovathi, M.D., HOD of O & G.
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7. Dr.S.R.Subramanian, M.D.,HOD of Medicine.
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10. Dr.T.Sundararajan, M.D., Associate Professor of Microbiology.
11. Dr.C.Kamalanathan, M.S., HOD of Orthopeadics.
12. Thiru.J.M.Arumugam, Legal Advisor, Salem.
13. Tmt.Ruby Thiagarajan, Social Worker.

MINUTES OF MEETING.

The Vice Principal, Govt.Mohan Kumaramangalam Medical College, Salem -30 has welcomed all the members of the Committee and explained the purpose of the Ethical Committee Meeting and asked the applicants to present their study before the committee.

The following Post Graduate students of this College have presented their study and requested Ethical Committee clearance for submitting their Dissertation to the Tamil Nadu Dr.M.G.R.Medical University, Chennai.

DEPARTMENT OF ORTHOPAEDICS.

1.Dr.A.Guruprasath, Ist Year Post Graduate student of M.S.(ORTHOPAEDICS), Govt.Mohan Kumaramangalam Medical College, Salem has presented his Dissertation on " FUNCTIONAL OUTCOME OF SERIAL CAST CORRECTION OF CONGENITAL TALIPES EQUINOVARUS BY PONSETI METHOD" under the guidance of Prof.Dr.C.Kamalanathan, M.S, HOD of Orthopaedics of this College.

2.Dr.R.Mugundan, IInd Year Post Graduate student of M.S.(ORTHOPAEDICS), Govt.Mohan Kumaramangalam Medical College, Salem has presented his Dissertation on " A PROSEPCTIVE STUDY OF CLINICAL OUTCOME OF TRAUMATIC DORSOLUMBAR FRACTURES TREATED WITH POSTERIOR STABILISATION WITH PEDICLE SCREWS AND FUSION BY MOSS MIAMI SYSTEM" under the guidance of Dr.A.D.Sampath Kumara, M.S, Associate Professor of Orthopaedics of this College.

3. Dr.R.Subramani, IInd Year Post Graduate student of M.S.(ORTHOPAEDICS), Govt.Mohan Kumaramangalam Medical College, Salem has presented his Dissertation on " A PROSEPCTIVE STUDY OF FUNCTIONAL OUTCOME OF BICONDYLAR TIBIAL PLATEAU FRACTURE MANAGED WITH LOCKING COMPRESSION PLATE" under the guidance of Dr.R.T.Parthasarathy, M.S, Associate Professor of Orthopaedics of this College.

out patient departments of Medicine, Peadiatrics, Dermatology and Psychiatry if this College Hospital and requested Ethical clearance to carry out the study.

2. Dr.N.Preetha,M.D., Assistant Professor of Pharmacology of this College has presented her study on "A PROSPECTIVE ANALYSIS OF ANTIBIOTIC PRESCRIBING PATTERN AMONG IN-PATIENTS IN A TERTIARY CARE HOSPITAL" in the Hospital premises.

The Ethical Committee examined the study in detail and is pleased to accord Ethical Clearance for the above two Assistant Professors of Pharmacology to carry out the study without affecting their normal duties.

Further, the committee has disapproved the following study presented by the Post Graduate Students of this College.

1. Dr.G.Jasmine, IInd Year Post Graduate student of M.S.(General Surgery), Govt.Mohan Kumaramangalam Medical College, Salem has presented her Dissertation on " PROSPECTIVE STUDY OF 50 CASES ON ETIOLOGY AND PATHOLOGY OF CERTIVAL LYMPH NODES BY FNAC AND BIOPSY IN GMKMCH, SALEM" under the guidance of Prof.Dr.A.Nirmala.M.S, Associate Professor of General Surgery of this College.
2. Dr.Mohamed Yasid. IInd Year Post Graduate student of M.D.(General MEDICINE), Govt.Mohan Kumaramangalam Medical College, Salem has presented his Dissertation on " PULMONDRY FUNCTION TEST IN TYPO-Z DIABETES"

DEAN.

To

1. All the Post Graduate Students – Concerned through the HOD of respective departments.
2. Dr.B.Keerthika,M.D., Assistant Professor of Pharmacology of this College.
3. Dr.N.Preetha, M.D., Assistant Professor of Pharmacology of this College.

Copy to:

1. All the Members of the Committee.
2. The Vice Principal, Govt.Mohan Kumaramangalam Medical College, Salem 30.

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ABSTRACT

Background: Clubfoot occurs in approximately one in 1000 live births and is one of the most common congenital birth defects. There have been many reports of successful treatment of idiopathic clubfoot with the Ponseti method in the western world; similar studies in developing countries like India are few.

Objective: To analyse the Functional outcome of serial cast correction of congenital talipes equinovarus by ponseti method.

Materials and Methods: The study is a prospective study, done at our "CTEV Clinic" conducted at Govt. Mohan Kumaramangalam Medical College, Salem, from July 2013 to september 2014. All patients were treated by serial manipulation and casting as described by Ponseti.

Results: In our series, we have treated 38 babies with idiopathic clubfoot by ponseti method by serial casting. Of the 38 babies 22 had bilateral affection and 16 had unilateral. 26 of the babies were male and 12 were female. We had recurrence of fore foot adduction in 6 of our patients(15.7%). Minor complications were noted in 13.15% of our cases.

Conclusion: We conclude that the Ponseti method is a very safe, efficient and economical treatment for the correction of club foot that radically decreases the need for extensive corrective surgeries especially in developing countries.

INTRODUCTION

Vast number of children are born with congenital clubfoot every year. Incidence of CTEV being one per 1000 live births. Most of these are kids born in countries where they remain untreated or poorly treated reducing their quality of life.

CTEV has been existent and known since time immemorial to mankind and so are the controversies it carries within itself. Many research has been done on these subjects and they all have contributed understanding the pathoanatomy and deciding upon the appropriate treatment. Still the literature states that treatment of club-foot is in general one of unvarying success.

Earliest written records of clubfoot management is found in the works of Hippocrates around 400 BC. Hippocrates was the first to advocate Orthopaedic management of club-foot by gentle manipulation and bandaging.

Nicholas Andry (1743) in his work"Orthopaedia" described the deformity as Pedes equines simulating the hoof of a horse.

In1932, Dr. H.Kite, recognized the fact that using force for manipulating CTEVor radical operative soft tissue release procedures are detrimental and, recommended moulding and casting in the corrected position for the non operative management of CTEV.

The literature from about 1970's to 1990's contains enthusiastic reports on the correction of congenital CTEV through extensive surgical release procedures. Over time, recognizing the complications of such procedures including recurrence, overcorrection, stiffness, and pain, there seems to be a renewed interest in conservative techniques for management of congenital clubfoot.

"Robert Jones discussing the treatment of clubfoot, rightly insisted the goal may be reached successfully by different routes" [3]

The management of club foot has evolved with two broad ideas over the last century. The first is the general acceptance of the principles of manipulation, strapping and serial plaster correction and the other one favours numerous surgical procedures for the correction of clubfoot. But none of the described method was able to achieve the goal of functional, painless and morphologically normal foot.

Way back in 1960s Dr. Ignacio Ponseti devised his method of conservative management of CTEV which starts from day one of age and is based on the fundamentals of kinematics and pathoanatomy of the deformity and successfully realigns clubfoot in infants without extensive and major surgeries.Ponseti'smethod has correct biomechanical basis for realigning deformed ankle and foot joints and corrects deformity due to the properties of soft tissue structures to undergo stretching utilizing the 'crimp'

Now there is consensus that the initial management of CTEV should always be non-surgical, starting from day one of life when the deformity can be easily corrected to achieve a plantigrade foot at earliest and it gives better functional & cosmetic results. So at present the mainstay in management of clubfoot is to diagnose the deformity as soon as possible and then to deal with the deformity as early as possible to realign the foot biomechanically . The involvement of the parents and their education regarding the disease is another important but often neglected aspect in achieving successful results.

AIM OF THE STUDY

To analyse the Functional outcome of serial cast correction of congenital talipes equinovarus by ponseti method.

REVIEW OF LITERATURE

The first classic description of Clubfoot was given by Hippocrates(460-377 B.C.) He thought that foetal intra-uterine mal-position was the cause of this deformity. He outlined the basic pathology about the deformity and outlined its management as follows. He emphasized that the treatment should be started as early as possible, gentle repeated manipulation is needed. Restoring the foot to its normal position was not sufficient and one should aim at over correcting the deformity. Correction should be maintained for at least 20 yrs of age.

M. Guerin (1838) was the first surgeon who reported the use of Plaster of Paris for Clubfoot.

Steindler (1920) advocated stripping of contracted plantar fascia, origin of Abductor hallucis, intrinsic toe flexors subperiosteally from plantar surface of calcaneum for severe cavus of the foot.

Lambrinudi (1927) removed a wedge of bone from the plantar and distal part of talus so as to maintain complete plantar flexion.

H.Kite (1930) popularized non operative treatment with serial manipulation and plaster cast immobilization. Immediate post-natal manipulation is initiated in Kite's method. It was derived from the

concept of three-point pressure. The pressure being given over the calcaneo-cuboid joint. The forefoot is manipulated with the heel stabilized with the calcaneo-cuboid joint as the fulcrum. Followed by casting to correct all deformities and maintaining in Denis Browne bar

Denis Browne (1934) devised a splint for maintenance of clubfoot correction obtained after manipulation.[1,5]

Dwyer's reported osteotomy of calcaneum for varus deformity. He elaborated an open wedge osteotomy of calcaneum.. He also performed lengthening of tendoachilles along with calcaneal osteotomy.[13]

Turco V.J. (1971) described clubfoot to be an exaggeration of normal equinus with subluxation of talonavicular joint. Turco also described single stage surgical correction of recurrent or resistant CTEV. Turco described one stage postero-medial soft tissue release, plantar and subtalar soft tissue releases and abnormally placed navicular and calcaneum are fixed in position with K wires. [18]

In 1980 Sterling J. Laav Veg and Ignacio V. Ponseti studied 70 patients(104 foot) that were treated at their hospital by serial cast correction and were followed for 10-27 years. They had satisfactory results in 88 % cases.[21]

A. Victoria Diaz and J. Victoria (1982) described the development of idiopathic CTEV. According to them, during development the foot progresses through three stages

(i) initial position

(ii) embryonic position

(iii)foetal position.

Morphological and Bony / soft tissue changes of the foot from “initial Stage” to the “embryonic stage” results due to growth of lower end of fibula and skeletal elements of lateral foot during "Fibular phase" of rapid growth. The structural changes from embryonic stage to foetal position is attributed to growth of the lower end of tibia and of the bones of medial side of foot. So based on the phase of the embryo , growth arrest of the foot occurs in an attitude closely resembling the “embryonic position”. If I the arrest occurs near the end of the “fibular phase” or inthe initial part of tibial phase, severe clubfoot may result.[22]

Douglas McKay (1982-3) in his series of about 100 patients performed surgery in about 55 fresh cases of clubfoot. He concluded that early motion by use of hinge cast increased the degree of ankle motion and that surgery was best performed before the child was 1 year old.[23]McKay single stage subtalar release was in fact a circumferential

soft tissue release of the posterior aspect , medial aspect, lateral aspect and plantar aspects of the foot. His patients had results better than those treated by posteromedial release with lesser wound complications, better range of motion and better correction.

B.B. Joshi (1988) devised his own indigenous apparatus called Joshi's External Stabilising System, (JESS), which works on the principle of controlled differential distraction for correction of clubfoot.[30]He was able to achieve a plantigrade foot in most cases by stretching the soft tissues differentially. The efficacy of this method in cases of neglected and severe cases of CTEV has been proved beyond doubt.[30]

Pirani et al (1995) outlined a method of assessing clubfoot using well-described simple clinical signs. He recorded an almost perfect inter observer reliability for his scoring system.[36, 37.]

Flynn et al (1998) conducted an independent study of two commonly used clubfoot classifications - Pirani and Dimeglio .Correlation coefficients were 0.901, p=0.0001 for the Pirani classification and 0.83 (p=0.0001) for the Dimeglio classification. On analysis, both Pirani and Dimeglio system of classification had good inter observer reliability. [41]

AETIOLOGY OF CTEV

Much has been said and written about the etiology of CTEV but the exact etiology is controversial till date..Through the years, many theories have been proposed and discarded. The theories have been rediscovered and represented with renewed interest by succeeding authors, during the past 200 years, the same basic concepts of etiology, with slight modification have enjoyed temporary acceptance as the solution to the unsolved puzzle -TURCO.

Aetiology of talipes equino varus deformity can be broadly classified into

A.IDIOPATHIC CLUBFOOT: When deformity is not associated with any other abnormalities in the body. This is the most common type

B.NON IDIOPATHIC CLUBFOOT: Where the deformity is a local manifestation of systemic musculo skeletal disorder.

IDIOPATHIC CLUBFOOT

Many theories have been put forward to explain the etiology of congenital talipes equinovarus. They are as follows.

1. Mechanical factors in utero

2. Germplasm defect

3. Arrested embryonal development

4. Heredity

5. Heredity and environment combined

6. Neuromuscular defect

7. Teratogenic theory

8.Vascular theory

All the above usually have no syndromic association or associated findings

1. MECHANICAL FACTORS IN UTERO

It is the oldest theory. Both Hippocrates (400 B.C) and Galen (200 A.D.) considered clubfoot to be caused by extrinsic pressure on fetus in utero. The proponents of this theory maintain that lack uterine liquor (oligohydramnios) prevents fetal movements and makes the fetus vulnerable to extrinsic pressures. Those opposing this theory point out that, during the 1st month of pregnancy when fetus is forming, it is floating in amniotic fluid, and there is no increased incidence clubfoot in pregnancies with an overcrowded uterus like in twin pregnancy, large babies, primiparous uterus etc. However, the possibility of transient elevation of uterine pressure at a vulnerable time could interfere with the development of the foot.

2. PRIMARY GERMPLASM DEFECT [10]

lrani and Shennan (JBJS 45A 1963) have suggested that the deformity probably results from a germ plasm defect involving the head of talus and neck of talus. They found that the neck of talus is always short and head facing medially and plantar wards. Since the anlagen for the talus is fully formed at 6 weeks and the tarsi well developed at 7 weeks, the changes must occur well before 7 weeks. Moreover a

completely normal shaped head and neck of talus fails to develop even on correction of the deformity,supporting this theory.

Antagonists of this theory say that the changes are secondary in nature. They argue that it is difficult to explain the germplasm defect occurring in one foot only and not as a bilateral defect.

3. ARRESTED EMBRYONAL DEVELOPMENT[10,11]

Heuter and Volkmann in 1963 first proposed arrest of foetal development as a cause of clubfoot. According to them, the deformity is due to arrest of physiological development of foot in early embryonic life. This is strengthened by the fact that the physiological position of foot in embryonic life is similar to talipes equinovarus.

Talipes equinovarus resembles the embryonic foot of 2 months age. A marvelous study of the evolution of the normal foot and the clubfoot was done by Bohm in 1929. He was able to show that variation seen in clubfoot relates to physiological positions that can be seen in the normal embryonal development of the foot, very convincing evidence of embryonal arrest theory.

Victoria Diaz in 1976 made a study in embryonic feet. The conclusion of the study was that changes in the position of embryonic

feet, as well as movements of the talus and calcaneus are due to a spurting growth of lower end of tibia and the fibula.

In the first stage (FIBULAR PHASE), the calcaneus was pushed and displaced into equinovarus position.

In the second phase (TIBIALPHASE) the talus was pushed and foot pronated into the usual fetal position and without tibial growth spurt the foot remained in equinovarus position. The additional circumstantial support is that in many clubfeet the medial malleolus have underdeveloped and is less prominent.

4. HEREDITY[13,14,15]

Studies by Palmer (JBJS 46A 1964) and Wynne Davies (1964) have shown that clubfoot deformity occurs much more frequently in those families that already have a member with a clubfoot. It is now believed that congenital Talipes Equino Varus is inherited by polygenic multi factorial trait probably in the form of an autosomal dominant inheritance

5. HEREDITARY AND ENVIRONMENT COMBINED

It is a theory of multi factorial origin. The combined hypothesis maintains that some intrauterine factor in conjunction with hereditary predisposition causes a disturbance in development at a crucial stage of

the embryonic development of the foot, thereby causing an arrest of normal fetal development.

6. NEUROMUSCULAR DEFECT[10,11,13]

Histo-chemical and electron microscopic studies of 60 clubfeet in patients with less than 5 years of age by Isacetal., indicated a dominant neurogenic factor in the causation and they were of the opinion that muscle imbalance may produce the deformity.On the contrary, Conaseco et al in 1974 did biochemical and electron microscopic studies on gastrocnemius and concluded “fibrosis in severe idiopathic clubfeet maybe a major factor in maintaining the deformity but should not be considered as a primary etiological factor.

There are many factors to show that the neurogenic cause is unlikely in development of clubfeet -

- Not all children with spinal bifida had clubfeet
- Neurologically deformed foot is usually more flexible than the congenital idiopathic foot.
- Neurological problems lead to deformities like plantar flexed talus and talipes Calcaneovalgus rather than talipes equinovarus.
- Neuromuscular deformities can be stretched and respond temporarily to manipulation unlike the majority of clubfoot.

7. TERATOGENIC THEORY[16]

In 1982, J Misawa et al observed malformations such as cleft palate, club foot and micrognathia in infants of Göttingen miniature pig which were fed with high dose of pyrimethamine

8.VASCULAR THEORY [81]

The vascular theory states that there is hypoplasia or absence of anterior tibial artery and dorsalis pedis arteries causing the talar deformity, as the anterior tibial artery is the prime blood supply for the talus.

NON-IDIOPATHIC CLUBFOOT

This type of clubfoot is secondary to other musculoskeletal abnormalities. A child may be born with clubfoot but the presence of other stigmata of skeletal disorder readily distinguishes the non-idiopathic deformity.

Clubfoot of non-idiopathic variety is associated with innumerable skeletal syndromes,

1. Arthrogryposis multiplex congenita

2. Nail patella syndrome

3. Congenital constriction bands

4. Neurological disorder, muscular dystrophies

5. Lead poisoning

6. Dystrophic dwarfism, teratogenic anomalies

7. Gordon syndrome

8. Mobius syndrome, Larsen’s syndrome, Smith and Lemliptiz syndrome,

9. Pierre Robin syndrome

It is usually associated with other abnormalities like supernumerary digits, eye abnormalities, cleft palate, and micrognathia, delayed motor and mental development.

1. ARTHROGRYPOSIS MULTIPLEX CONGENITA[17]

This is characterized by muscle wasting, rigid joint contractures and high incidence of clubfoot. A common triad is talipes equinovarus, hyperextension knee contracture and the congenital dislocation of hip joint. The clubfoot here is a rigid deformity resistant to all forms of treatment.

Manipulation is started as early as possible. When hip, knee and feet is involved, a hip spica is applied. Results of surgery are poor and multiple surgeries are required to achieve a plantigrade foot. Initially a soft tissue surgery is tried. Bony surgery includes naviculectomy, talectomy, metatarsal and calcaneal osteotomies.

2. HEREDITARY ONYCHO OSTEODYSPLASIA[18]

Also known as nail patella syndrome or Fong disease, it is a hereditary disorder of ectodermal and mesodermal tissues and is characterized by the absence of patella, nail abnormalities and presence of subluxation of head of radius. Nails may be absent or have vertical grooves. According to Match (New York State Medical Journal 1973), 4 to 8 patients had clubfoot with mild deformities, which could be corrected by manipulations.

3. CONGENITAL CONSTRICTION BANDS[91]

It is characterized by circumferential constriction bands (STREETER DYSPLASIA). it is a rare deformity, which involves both upper and lower limbs. The clubfoot in this condition is associated with congenital amputations of toes,edema and vascular impairment. Before the clubfoot correction, 'Z' plasty should be done to remove the constriction bands and improve the circulation.

4. NEUROLOGICAL DISORDERS[20]

Clubfoot is associated with conditions like meningomyelocele, spina bifida, spinal cord defect, hydrocephalus, cerebral palsy and other ill-defined disorders. One should suspect this group when the deformities are asymmetric. E.g Talipes calcaneovalgus on one side and talipes equinovarus on other side.

5. MUSCLE IMBALANCES[21]

Clubfoot due to myopathies may be subtle. The deformity can be easily corrected by manipulation. A chance of over correction may be

there with surgery. Surgery is usually delayed, and is only advocated when necessary.

6. GROSS CHROMOSOMAL ABNORMALITIES[22]

A recurrent chromosome 17q23.1q23.2 microduplication is found in a study to be associated with clubfoot.

PATHO-ANATOMY

Club foots have predominant involvement of ankle and foot. The exact nature of the deformity in clubfoot is still controversial. Most authors consider the principal deformity to be a congenital dislocation of the talo-navicular joint, but it may be possibly better considered to be a fixed exaggeration of the normal equino varus position.

The major deformity is believed to be an inward rotation of the whole foot upon the talus. This rotation takes place primarily at the talo-calcaneonavicular joint but also at the calcaneocuboid joint.[4]

Nowadays, most of the workers believe that the soft tissue abnormality is the main cause of the deformities in clubfoot and that the bony changes occur secondary to the soft tissue abnormalities.[4]

THE DEFORMITIES IN CLUBFOOT

The typical clubfoot consists of a deformed foot in equinus, varus, adduction and in some cases a cavus component. Varying degrees of severity can be seen in the newborn, in addition to varying degree of associated rigidity.

Some feet which are extremely rigid, are usually smaller, stubby with a short first metatarsal ray. Feet that are less rigid and more pliable on manipulation are usually longer than those with rigid deformities.

Deformities that can be almost fully corrected on initial manipulation should be considered mild positional deformities. Equinus deformity of the foot is accompanied by an inversion of the heel, adduction and varus of the forefoot.

The medial border of the foot is concave and elevated and its planter surface faces upwards; the lateral border of the foot is convex and depressed downwards. The posterior tuberosity of the heel is pulled upward, inverted, difficult to palpate and less visible. The older child may have a callosity on the dorsal aspect of the fifth metatarsal. The bony prominence visible and palpable on the dorsal and lateral side of foot represents the talar head and neck, which are partially uncovered because the navicular and the calcaneum have been displaced medially. [4,15]

Skin Abnormalities:

The skin over the dorsal and outer aspect of foot is usually stretched out, thin and atrophied. Some feet have a deep cleft on the medial plantar surface: usually they have a severe cavus deformity with a fore foot contracture.

Some feet that are rigid and have a severe equinovarus deformity also have a single deep cleft in the skin just above the heel and the prominence of the heel is obscured. The skin along the medial aspect of the foot below the medial malleolus is contracted and 'notorious poorly nourished' an important consideration in surgical treatment.[4,15]

The knee and lower leg:

At birth the knee appears normal with the usual knee flexion contracture. A hyperextension at the knee becomes evident later as a consequence of the fixed equinus deformity of the foot. Genu valgum is commoner in the older child with a severe uncorrected bilateral equinovarus deformity: this is a compensatory acquired adaptation as the child attempts to keep the more involved foot in a plantigrade position.[4,15]

THE ANKLE:

In the normal foot, the ankle mortise faces slightly laterally. In the clubfoot this external rotation of the mortise is increased. In resistant feet, this lateral orientation of the tibio fibular unit increases with age. Another factor is the child's attempt to compensate for the varus adduction deformity of the foot by rotating the leg externally on weight bearing and walking.

The lateral malleolus is palpable posteriorly, which is to be expected with the increased external rotation of the mortise. The medial malleolus is usually underdeveloped and appears to be anterior to normal.

The deformities are equinus, adduction and inversion of the hind foot and adduction and inversion of the forefoot. As the child grows adaptive, changes occur in the bones and soft tissues because of abnormal position. Bony changes are secondary and predictable.

EQUINUS:

The talus is plantar flexed so that a large portion of superior articular surface is out of the ankle mortise and the head is prominent and easily palpable, dorsally. The calcaneum is also plantar flexed and this accounts for the high heel. The equinus of the fore foot occurs at transverse tarsal joint and it accounts for the peculiar “slapping gait” of the partially corrected clubfoot where fore foot touches the ground before the hind foot.

ADDUCTION:

The calcaneum is adducted so that it rolls under the talus. The forefoot adduction is present at the mid tarsal joint and is often the most persistent deformity. This and the adduction at metatarso phalangeal joints result in “pigeon toed gait”.The forefoot adduction should be corrected only after aligning the forefoot with hindfoot, after cavus correction. This is the most common deformity to recur after full correction.

INVERSION:

The talus is fixed between the malleoli and cannot be inverted but the calcaneum is inverted so that sustentaculum tali approaches the medial malleolus. The forefoot is also inverted at the transverse tarsal joint. It is a combination of fore foot and hind foot inversion, which results in supination of the fore foot.

CAVUS:

The fore foot plantar flexion causes a cavus deformity and contributes to the composite equinus.

TIBIAL TORSION:

Tibial torsion may be defined as any turning round of tibia along its length, which results in a change in orientation of the upper and lower articular surface of tibia. The possible rotational abnormalities are, internal and external. According to Stewart, although the tibial torsion is not a cause of clubfoot it tends to perpetuate the deformity by causing varus bending of the foot at tarsometatarsal joints in walking.

Tibial torsion exists in two forms.

1. True tibial torsion and

2. Pseudotibial torsion

TRUE TIBIAL TORSION:

The bones, which are fixed, undergo torsion, like tibia and fibula. Tarsal and metatarsals, which are movable to some extent, will not undergo any torsion but get displaced.

PSEUDO-TIB1AL TORSION:

Pseudotorsion of tibia is apparently ligamentous affection and is distinguished by the following test. The child is laid in prone position and the knees are flexed. On examining the rotations of the leg, there is an increased internal rotation when compared to external rotation in pseudo torsion. This can be explained as due to the ligamentous affections. However, in true torsion there should not be any unequal limitation of rotations.

Osseous deformities:-

Many investigators have observed that the overall size of all tarsal bones is smaller in the clubfoot than in normal foot thus producing asymmetric size in a unilateral deformity. Both legs are usually equal in length. [15]

The talus:-

While the talus is the least displaced, it undergoes the most severe and consistent changes in form. The talus has no muscle attachments and is passively forced into equinus by its articulations and attachments to the calcaneum and navicular.[15]

Body of the talus:-

In the equinus position, only the posterior half of the trochlea articulates with the tibia; the forward portion of the trochlea is out of the mortise anteriorly. In a clubfoot the anterior wider portion of the body probably never enters the ankle joint, therefore this portion of the trochlea never had the opportunity to respond to physiological stress. As a consequence, the anterior trochlea is prone to develop the adaptive morphologic changes.[15]

Neck of the talus:-

The most important constant distortion is found in the neck and head of talus. Normally, the long axis of the neck and the head of the talus is directed slightly medially in relation to the body (about 150 degrees) in clubfoot the medial deviation if the neck and head is increased to form a more acute angle with the axis of the talar body; the degree of talar deviation is quite variable (115 to 135 degrees).

In addition, the neck is foreshortened and the usual constriction of the neck is absent. This heaping up of bone in this part of the trochlea and

talar neck , and the medial deviation of the talar neck, form a bony mass that impinges on the anterior lip of the tibia in dorsiflexion; thus the entrance of the talus into the mortise is impeded, contributing to the equinus deformity.[15]

Talar Head:-

The round shaped talar head normally faces forwards and is covered by concave surface of the navicular. In the clubfoot, the Talar head and the facet for the navicular bone face medially, the talonavicular articulation is oriented in a more sagittal plane compared to the normal coronal orientation. The head of the talus is usually broader than normal with varying degrees of distortion. Correlating the talar head deformity with prior treatment suggests that some of the distortion may be attributed to iatrogenic compression of the cartilaginous anlagen by manipulative treatment.[15]

CLASSIFICATION OF C.T.E.V [9]

Classification according to treatment stage:

- **Untreated clubfoot**: Affected child under 2 years of age and has had no treatment till date.

- **Neglected clubfoot**: affected child more than 2 yrs and has had no treatment till date.

- **Treated clubfoot**: Club foots that have completed the casting phase of Ponseti method.

- **Recurrent clubfoot**: recurrence of deformities (equines and supination)after initial good correction.

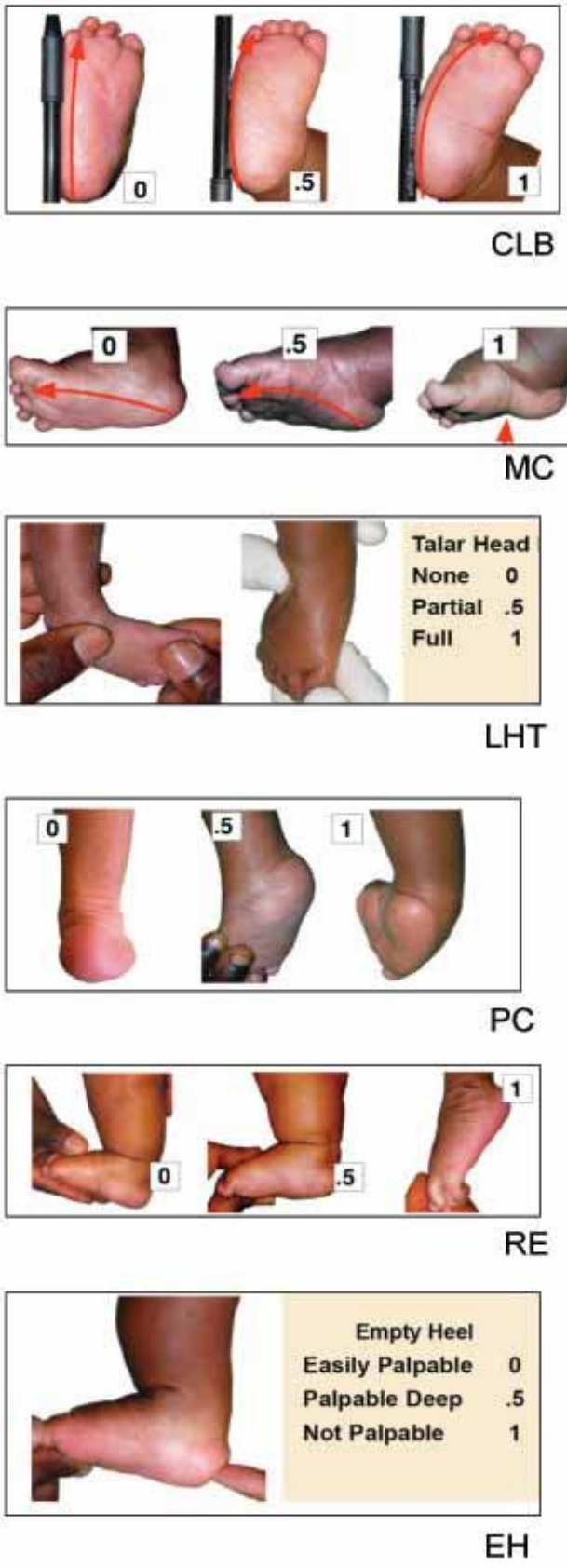
- **Resistant Clubfoot**: Previously untreated club foot that does not correct with ponseti technique.

- **Complex clubfoot**: any club foot that has received any form of treatment other than ponseti.

Analysis of results of clubfoot management is affected by absence of a common classifying methodology for analyzing the extent of the involvement and the post treatment results. Many classification have been proposed, like those by Carroll, Goldner, and Catterall. The more recent classifications by Pirani etal. and Diméglio . are purely dependent on Clinical findings and do not require xray measurements or special tests.

PIRANI SCORING SYSTEM[23,24]

Dr. Pirani has developed a consistent and convincing method of clinically evaluating the magnititude of foot and ankle morphological changes in a fresh case of clubfoot less than 2 years of age. Recording the extent of involvement allows the treating surgeon to asses where he or she is on the roadmap of management, asses the need for tenotomy, and to inform and reassure parents about the progress in management. It allows reliable comparison of results. The Pirani method measures six clinical parameters. These parameters are graded as normal, moderately abnormal, or severely abnormal and scored as 0, 0.5, 1 respectively.



THE PIRANI SCORING SYSTEM

Midfoot score

The following three components are included in the Mid Foot Score (MFS), for grading the extent of involvement

1.Curved lateral border

2.Medial crease

3.Coverage of the talar head

Hindfoot score

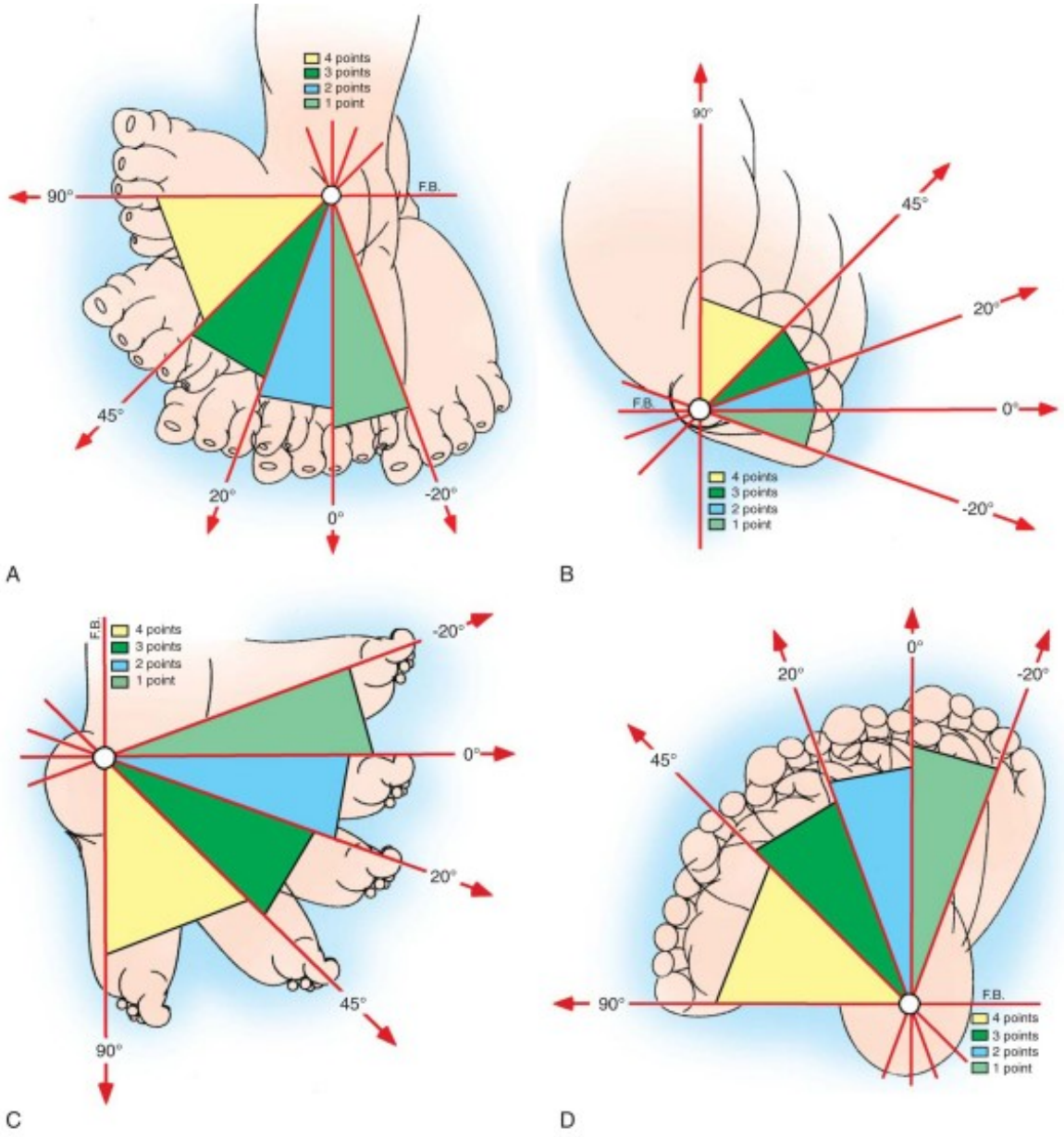
The following three components are included in the Hind Foot Score (HFS), for grading the extent of involvement

1.Posterior crease

2.Rigid equinus

3.Empty heel

DIMEGLIO SCORING SYSTEM[25]



DIMEGLIO SCORING SYSTEM[25]

Four clinical signs are evaluated after gentle corrective manipulation and documented using a hand held goniometer

Parameters Measured	Reducibility (degrees)	Score
Equinus deviation in sagittal plane (Fig. 26-30A)	90 to 45	4
Varus deviation in frontal plane (Fig. 26-30B)	45 to 20	3
Derotation of calcaneopedal block in horizontal plane (Fig. 26-30C)	29 to 0	2
Adduction of forefoot relative to hindfoot in horizontal plane (Fig. 26-30D)	0 to −20	1
	<−20	0
		16
Other elements considered		
Posterior crease marked		1
Mediotarsal crease marked		1
Plantar retraction or cavus		1
Poor muscle condition		1
Possible total score		*20*

Grade	Type	Frequency (%)	Score	Reducibility
I	Benign	20	1-4	>90% soft-soft, resolving
II	Moderate	33	5-9	>50% soft-stiff, reducible, partially resistant
III	Severe	35	10-14	>50% stiff-soft, resistant, partially reducible
IV	Very severe	12	15-20	<10% stiff-stiff, resistant

THE PONSETI METHOD

Ponseti'sMethod of clubfoot correction, "Aims at biomechanical realignment rather than anatomical or radiological correction"

Scientific Basis of Management[4]

The Ponseti management of CTEV relies on the biology of the deformed foot and the functional foot anatomy.

Biology

Clubfoot is not a malformation of embryonic life. The developing foot Closely resembles a clubfoot between 12-24 weeks of gestation. Picking up a case of CTEV in the first trimester using USG is extremely difficult. Therefore, clubfoot is probably a developmental deformation of the foot. The shape of the tarsal joints are modified along with the bones of the tarsal joints. The forefoot is pronated, making the plantar arch more concave (The cavus deformity).

In the clubfoot, there appears to be excessive pull of the tibialis posterior. The bulk of the muscles of the involved foot are small and the involved muscles are also shorter in length and are comparatively smaller in size and length than the normal foot.

In CTEV, the ligaments of the posterior aspect and medial aspects of the ankle are thickened and under tension, which keeps the ankle in equinus and the calcaneum in adduction and inversion. There is an inverse relationship between the deformity in CTEV and bulk of the muscle in the involved leg..

A severely affected club foot may make the gastrosoleus appear as a tiny muscle in the proximal third of the calf. The tendency for abundant collagen production in the tendons,and muscles might persist until the child reaches 4 years and may lead to relapses. Abundance of collagen and cells in the tendons and muscles can be visualized under the microscope. The collagen bundles display a wavy appearance called as ‘crimp’. “The crimp” permits the ligaments and tendons for stretching.The crimp usually reappears in few days time, so that further stretching is possible. This explains the basis for manual correction of the deformity by manipulation and serial casting.

Kinematics

For achieving adequate correction of the various deformities in club foot a thorough knowledge of anatomy and the function of the foot is essential.

Unfortunately, many orthopaedic surgeons managing club foot are of the idea that the subtalar and Chopart joints together have an axis of rotation which passes from anteromedial superior to posterolateral inferior, traversing through the sinus tarsi. Many believed that heel varus can be corrected utilising this imaginary line as the fulcrum for Pronating the foot. But pronation along this axis results in further cavus without actually correcting the deformity

In the clubfoot , the anterior portion of the calcaneus lies beneath the head of the talus. This position accounts for varus and equinus deformity of the heel. Abduction of the calcaneus to its normal relationship with the talus will correctthe heel varus deformity of the clubfoot. The clubfoot is a deformity primarily of the tarsal bones which are mostly cartilaginous, are usually in extreme flexion,adduction, and inversion at birth. The talus is severely plantar flexed. Talar neck is medially and plantarly deflected, and Talar head is wedge shaped. The

navicular bone is in extreme medial displacement and lies close to the medial malleolus, and is in articulation with the medial aspect of talar head. The calcaneum is adducted andinverted under the talus.

In clubfoot the calcaneus, the navicular and the cuboid are rotated medially in relation to the talus, and are held rigid in that position by the tight medial structures. Although the whole foot is in extreme supination, the forefoot is pronated in relation to the hind foot and this causes cavus, the first metatarsal being in more plantar flexion than the lateral metatarsals.

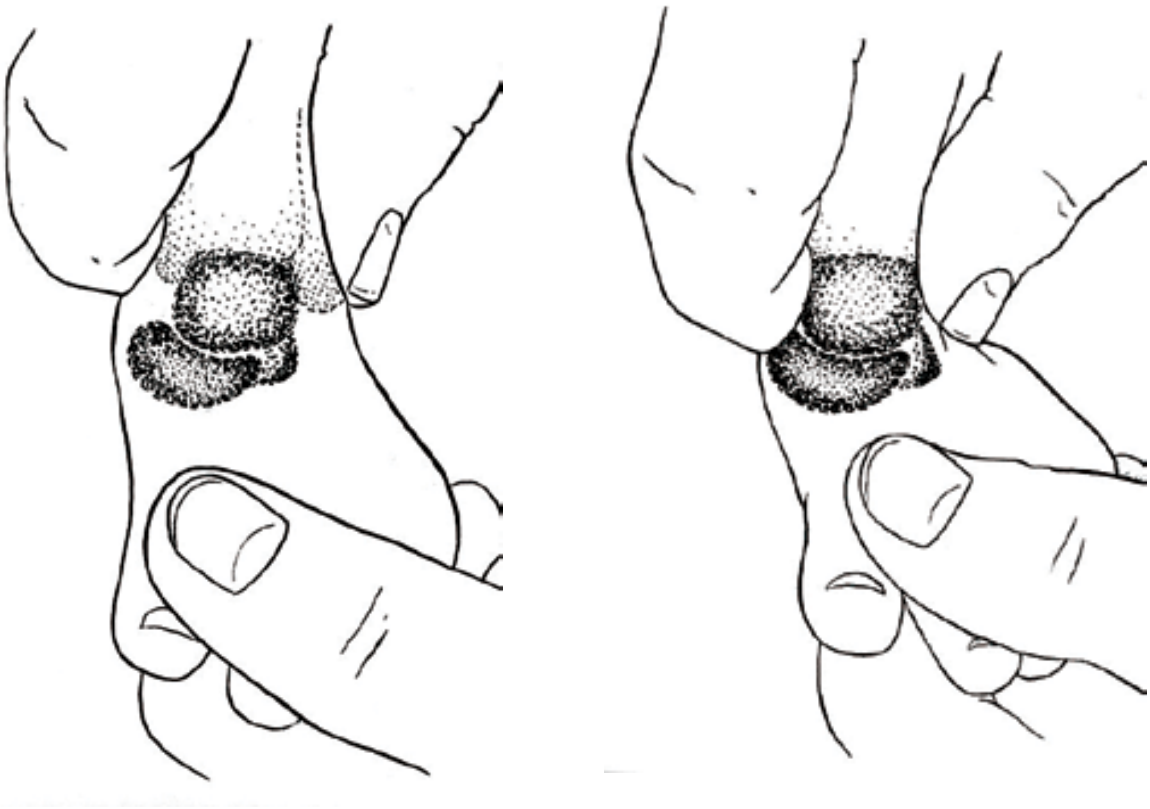
DETAILS OF THE PONSETI TECHNIQUE

Parental education and assurance are of prime importance in the success of this method. The baby is assessed from head to toe with special care to examine the spine and hips. The severity of the deformed foot is assessed using Pirani scoring system.

FIRST VISIT

Initial visit is always in the early neonatal period itself. Keep the infant in comfort throughout the visiting period. Feeding may be allowed

during the application of corrective cast. As far as possible Casting is done by the surgeon himself.



Manipulation requires abductive stretching of the forefoot under the stabilized talar head.

Reduce the cavus:

The high medial arch (cavus) , results from a pronated forefoot with respect to the hind foot..Cavus is usually supple in newborns and correction requires only supinating the forefoot by elevating first metatarsal to achieve a normal longitudinal arch of the foot. It is necessary to bring forefoot in the same plane as that of hindfoot, because only when this is achieved, the whole foot can be manipulated as a single unit keeping talus as the fulcrum.

Here it is important to remember that as the cavus is reduced, the deformity seems to be exaggerated and parents should be explained about the importance of getting the forefoot aligned to the hindfoot. Cast is

applied in this position and the parents are advised to return after 1 week. Possible cast complications have to be explained

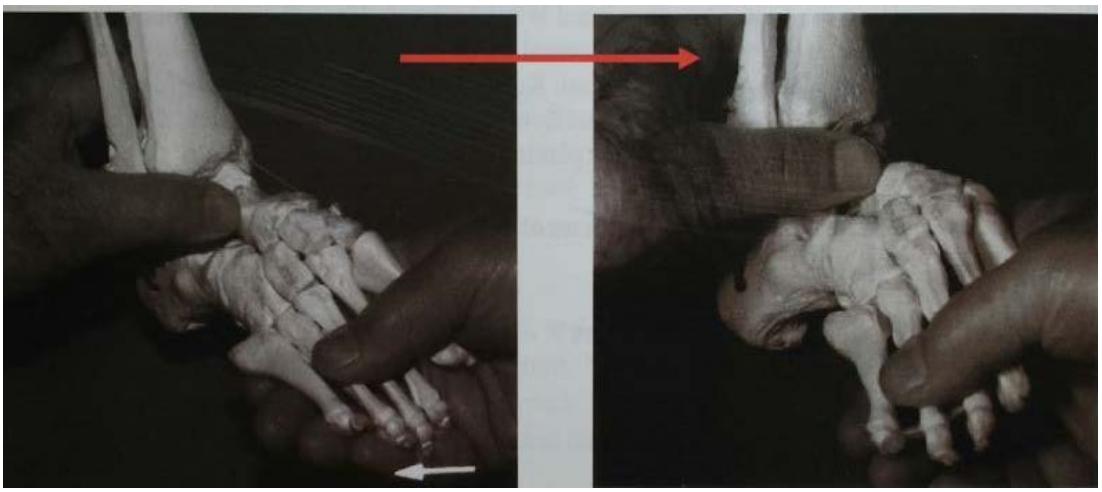
SUBSEQUENT VISITS

Cast is removed and the severity of the deformity is assessed. Cavus must be corrected by the second visit, if not the first step is repeated.

Exactly locate the head of the talus:

The lateral malleoli is palpated with thumb & index fingers of one hand Holding the toes with the other hand.. Next, slide your thumb and index finger forwards to palpate the head of the talus in front of the ankle

.Talar head is barely covered by the skin in front of the lateral malleolus. The anterior part of the calcaneus will be felt beneath the talar head.



On moving the supinated forefoot-laterally, you will be able to feel the navicular move ever so slightly in front of the head of the talus as the calcaneus moves laterally under the talar head.

Stabilize the talus:

The talar head is supported by the thumb. The ipsilateral index finger of the same hand that is stabilizing the talar head should be placed behind that lateral malleolus. This further stabilizes the ankle joint while the foot is abducted beneath it and avoids any tendency for the posterior calcaneal-fibular ligament to pull the fibula posteriorly during manipulation.

Next, on abducting the supinated fore foot stabilized by the thumb over the head of the talus, the foot is maximally abducted as far as can be done without causing much discomfort for the infant. The corrected position is held with mild pressure for about a . The lateral motion of the navicular and of the anterior part of the calcaneus increases as the clubfoot deformity corrects. Full correction should be possible after the fourth or fifth cast

The image after the fourth cast

Complete correction of the cavus, adductus, and varus can be achieved. Equinus though improved, is usually inadequate, necessitating a heel cord tenotomy. In very supple feet, equinus may be corrected by additiona corrective casts without tenotomy. When uncertain tenotomy may be done.

Image showing the sequence of shape change expected with each cast correction

DECISION TO PERFORM TENOTOMY

It has to be decided whether adequate correction of the deformity is achieved to do heel cord tenotomy to correct equinus and to complete the sequence. In other words, all the deformities except equinus have been fully corrected. This stage is achieved when the anterior part of calcaneum can be laterally rotated from undersurface of the talus. This

abduction permits to correct the equinus without pressing upon the talus between tibia and calcaneum. If necessary one or two additional casts can be performed to achieve correction of forefoot adduction prior to tenotomy.

The best sign of sufficient abduction is the ability to palpate the anterior process of the calcaneum as it abducts out from beneath the talus. Fore foot abduction of approximately 60 degrees with respect to the frontal plane of the tibia can be achieved. By this time as all the deformities except equinus have been corrected, midfoot pirani score should have fallen below 1 and hindfoot pirani score should still be over1.

Post-tenotomy Cast:

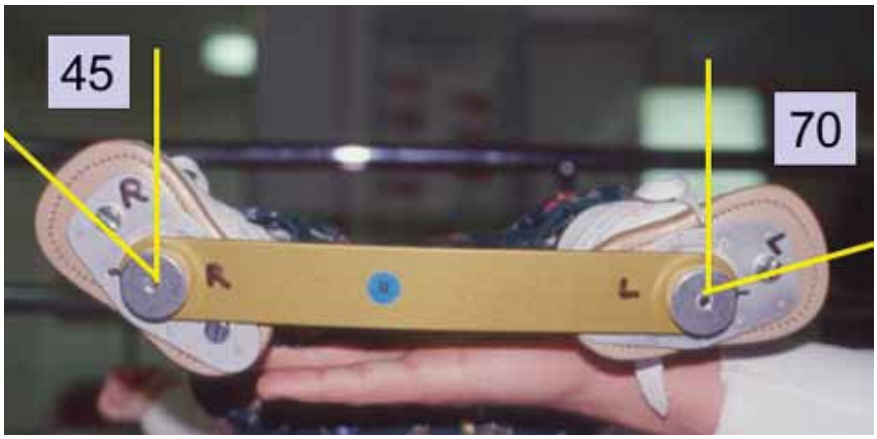
After tenotomy apply the final cast with the foot in 60 to70 degrees abduction, and 15 degree dorsiflexed. Thefoot now presents an over-corrected appearance. The Cast is continued for 3 weeks to maintain the correction. Consider cast change if it wears out before the 3 week

period. . analgesic or antibiotics are usually not mandatory. This is Probably the final cast in the management schedule.

Bracing:

Ponseti's method uses a brace to maintain the foot in abducted and dorsiflexed after the removal of the post-tenotomy cast.The brace is a bar attached to straight open-toed shoe.

FOOT ABDUCTION BRACE



This amount of foot abduction is required to maintain the abduction of the calcaneus and forefoot and to avoid relapse.The brace keeps the medial soft tissues stretched out once the child is out of the casting phase. The brace does not restrict the knee movements so that the child can extend both knees completely which stretchesthe gastrosoleus tendon. Abduction of the feet in the brace, combined with the slight bend,makes the foot to dorsiflex. This stretches the gastrocnemius muscle and heel-cord tendon.

Bracing Protocol:

Relapse rate can be more than 80% if bracing is not used for CTEV correction. The brace comprises of open-toed, high top, straight shoes attached to a bar . For unilateral cases, the brace is set at 60 to 70 degrees of external rotation for the involved side and at 45 degrees of external rotation for the unaffected side. In bilateral cases, the brace is set at 70 degrees of external rotation for both the affected sides. The bar must be of adequate length so that the heels of the shoes are at shoulder width. A common mistake is to recommend too short a bar, which makes the child uncomfortable. A narrow brace is a very common cause for a

lack of compliance. The bar should be bent 5 to 10 degrees with the convexity away from the child, to hold the feet in dorsiflexion.The brace should be worn full time (day and night) for the first 3 months after the last cast is removed.

After the first 3 months, the parents are instructed to make the child wear the brace for 12 hours at night and 2 to 4 hours in the middle of the day, for a total of 14 to 16 hours during every 24-hour period. This protocol continues until the child is 3 to 4 years of age. Brace usage may result in excessive heel valgus and external tibaial torsion. If that occurs,the surgeon must decrease the external rotation of the shoes on the bar from around 70 degrees to 40 degrees.

Common errors in correction of clubfoot according to Ponseti:[4]

Pronation of the foot which exaggerates the cavus and locks the adducted calcaneus under the talus, whereas the midfoot and forefoot are twisted into eversion.

External rotation of the foot to correct fore foot adduction while the calcaneus is still invarus. This results in a posterior transposition of the lateral malleolus by externally rotating the talus in the ankle mortise. The

posteriorly displaced lateral malleolus, seen in poorly treated clubfoot, is an iatrogenic deformity.

Abducting the foot at the midtarsal joints with the finger pressing on the outer side of foot near the calcaneo-cuboid joint **(Kite's major error).** By abducting the foot along with pressure over the calcaneo-cuboid joint the abduction of the calcaneus is prevented, thereby interfering with correction of the heel varus.

Attempts to obtain a perfect anatomical correction: It is a misconception that early re-alignment of the displaced tarsal bones results in anormal anatomy and good long term functional results in clubfoot. There have been many studies confirming that radiological findings does not correlate with the long time functional results. In a case of severe CTEV , thorough correction of the entirely medially displaced navicular may not be possible by manipulation alone as the medial tarsal ligaments are not amenable to stretching significantly.

Therefore in infants, the medial taut structures must be progressively stretched as much as they can yield rather than cut, without considering the ability to achieve a complete reduction.

Once the navicular displacement is corrected atleast partially, the forefoot can be adequately aligned with the the hind foot because the ligament in front of the navicular and the bifurcate ligament are stretchable, allowing fore foot abduction. The calcaneum can be abducted to bring the heel into a normal neutral position. This anatomical correction, though imperfect usually results in good appearance and functional result.This avoids many complications associated with surgical release.

Complications:

Manual cast correction done after manipulating with after adequate padding usually gives excellent results without any complications. The following are the complications to be expected and looked for,

A.Pressure Ulcers

B.Skin allergy

C.Slipping of cast

D.Circulation problems

E.Rocker bottom foot

F.Muscle atrophy

MATERIALS AND METHODS

This study was done at our "CTEV Clinic" conducted at Govt. Mohan Kumaramangalam Medical College, Salem. Study was conducted from July 2013 to september 2014.

Study design:-

The study is a prospective study,

Source of Data:-

All the children from birth to 12 months of age with congenital idiopathic clubfoot attending the CTEV Clinic from August 2013 to August 2014 at our hospital and who are willing to undergo treatment.

Inclusion criteria: –

1) Infant from birth to 12 months of age with clubfoot deformity

2) Infants with idiopathic clubfoot.

Exclusion criteria –

1) Infants with Non-idiopathic clubfoot like myelodysplasia , complex idiopathic clubfoot ,paralytic clubfoot.

2) Previously operated for clubfoot

3) Age more than 12 months

38 cases being selected from the registered patients in the "CTEV Clinic with untreated deformed foot and age at presentation less than 12 months.

Each patient was registered and detailed personal history was recorded including the age, sex, father's & mother's name, address, date of first reporting, age of reporting, detailed history of previous treatment, etc. A thorough general & local examination was carried out & the deformity was scored according to Pirani' s classification at each visit before applying cast.

The score was plotted against the time and the trend of score was noted with reference to effect of manipulations or other interventions on deformity.

Manipulations were done by Ponseti's method followed by corrective casts at weekly interval without anaesthesia. Depending upon the response of the deformity to serial casting as evident by improvement in Pirani Scoring since institution of treatment, the treatment was either continued or modifications were recommended. Patients were followed up weekly for corrective casting till tenotomy and corrective cast was applied for 3 weeks after final correction or percutaneous Tendo Achilles

tenotomy. We performed the tenotomy under anesthesia. Then the patients were advised regarding bracing with Dennis Browne splints for 3 months and followed-up to instruct regarding night time bracing for 3- 4 years. Modified CTEV shoes in children who had started bearing weight on lower limbs were given.

The Ponseti Technique[4]

The treatment is started as far as possible in the early neonatal period itself. The child should be kept comfortable through the casting process so that the casting can be done comfortably and perfectly.

Reduction of cavus:

The first aspect of serial cast correction is correction of high arch of the foot by aligning the fore foot to the hind foot perfectly. The high medial arch (cavus) , results from a pronated forefoot with respect to the hind foot..Cavus is usually supple in neonates and correction requires only supinating the forefoot by elevating first metatarsal to achieve a normal longitudinal arch of the foot. It is necessary to bring forefoot in the same plane as that of hindfoot, because only when this is achieved, the whole foot can be manipulated as a single unit keeping talus as the fulcrum.

The forefoot is supinated to the extent that visual inspection of the plantar surface of the foot reveals a normal appearing arch—neither too high nor too flat.

Manipulation:

The manipulation comprises of abduction of the foot below the stabilized head of talus. First the talar head is located. The heel varus and fore foot adduction are corrected simultaneously .To achieve this, the talar head is located ,and this serves as the fulcrum for correcting the deformity. The talar head is identified by palpating anteriorly from the lateral malleolus. Underneath the talar head the anterior part of calcaneum can also be identified As the foot is laterally rotated with the talar head stabilized, the movement of the navicular bone can also be assessed. The manipulated position is held with least possible pressure for about a minute and released. The foot in this sequence is not pronated at any stage

Second, third, and fourth casts:

The heel varus and fore foot adduction are progressively corrected through these stages. The equinus slowly corrects with correction of fore foot adduction and heel varus. This is due to the tendency of the calcaneum to dorsiflex under the talus. No attempt is made to correct the equinus by manipulation at this stage

Foot appearance after the fourth cast:

The fore foot adduction and heel varus will be corrected at this stage. Equinus though reduced is not adequate, for which a heel cord tenotomy is usually necessary. Sometimes in the very supple foot, equinus is managed with few additional cast corrections without tenotomy. If the progress is uncertain tenotomy is performed.

THE TECHNIQUE OF CAST APPLICATION:-

Manipulation before casting:

The foot is manipulated each time prior to application of the cast. The foot should be held by the toes. Holding the calcaneum is avoided to allow it to abduct along with foot abduction

Application of soft cotton roll padding:

A very thin layer of cast padding is applied around the foot after manipulation. Throughout the application of soft cotton roll around the leg the foot is held by the toes with the talar head stabilized with one finger.

Cast application:

Initially the cast is applied as a below knee cast and then converted into an above knee cast with knee in 90 degree flexion. The plaster is applied smoothly. Too much tension while applying the cast is avoided.. The Plaster is rolled over the surgeons finger also so that finally there will be adequate room for the toes to move about

Moulding the cast:

This is done using mild pressure. Continous pressure is best avoided over the talar head. The pressure is applied and relaxed alternatively good moulding is done to maintain the arch of the foot to prevent any possibility of rocker bottom foot. Both the malleoli are moulded. The entire process of moulding should be a dynamic one and

static pressure at any particular point is avoided as much as possible. The moulding process is continued till the plaster hardens.

Conversion to Above knee cast :

Adequate padding is given at the upper thigh to avoid skin irritation .The Plaster of Paris may be layered over the front of knee to reinforce the cast. The cast is finally trimmed to allow enough room for toes

Cast Removal:

The cast is removed in the subsequent visit at the CTEV clinic just before the application of new cast. Early cast removal is to be avoided as considerable correction may be lost in the period when the child is out of the plaster. Usually we use a plaster knife to cut the plaster.

Assessment of the need for tenotomy:-

A critical point in the treatment protocol is to decide whether adequate correction has been obtained to go ahead with the heel-cord tenotomy. This is assessed as the stage when the anterior calcaneus is abducted out under the talus This abduction allows the foot to be safely dorsiflexed without crushing the talus between the calcaneus and tibia. In uncertain

situations a further few castings can be done till the foot is in sufficient abduction to undergo the tenotomy.

Features of a well abducted foot:[4]

It is mandatory to verify that the foot is adequately abducted to bring the ankle into 0-5 degrees of dorsiflexion prior to tenotomy. This is best assessed by the ability to feel the anterior process of calcaneum under the talus.

The final outcome:

The end result should be foot over-corrected in abduction.. It is actually a full correction of the foot into maximum normal abduction. This helps prevent relapses.

Equinus Correction [4]

It is made certain the Pre-requisites forequinus correction have been met.

a) Pirani score for Mid foot contracture is 1 or less
b) Heel in valgus
c) Talar head measures 0
d) Foot in abduction

Percutaneous heel cord tenotomy: [4]

Skin preparation:

The foot is prepared thoroughly from midcalf till midfoot with an antiseptic the foot is held by the assistant from the toes with the fingers of one hand and the thigh is held with the other hand.

Anaesthesia :

We performed the tenotomy under general anaesthesia / local anaesthesia

Equipment:

No.11 or any other small blade is used.

Heel cord tenotomy:

The tenotomy is performed around 1.5 cm above the calcaneuum with

The assistant holding the foot in maximal dorsiflexion. A “pop” is felt as the tenotomy is completed. A further 20 to 25 degrees of dorsiflexion is usually gained after the Heel-cord tenotomy.

The final cast after tenotomy:

The final cast is applied with the foot in 60-70 degrees of abduction. After tenotomy the limb is immobilized in the above knee cast for 3 weeks

Removal of the cast:

At the end of third week the cast is removed. Thirty degrees of dorsiflexion should now be possible, and the surgical scar is minimal. This foot is now ready for brace application

PIRANI'S METHOD OF CLUBFOOT EVALUATION[36,37]:

Dr. Shafique Pirani, Clubfoot Clinic of Royal Columbian Hospital, University of British Columbia, Canada developed this valid, user friendly and reliable method of clinically evaluating the severity of a virgin club foot deformity.

He had identified 6 well described clinical signs of clubfoot. Three of these signs indicate primarily Hind Foot Contracture (HFC) and three signs indicate primarily Mid Foot Contracture (MFC).

The abnormal area on the involved foot is compared to normal side(if deformity is not bilateral) and scored:: -

0 = No deformity

0.5 = Moderate deformity

1 = Severe deformity

Hind Foot Contracture (HFC)

1. Posterior crease (PC)

2. Rigid Equinus(RE)

3. Empty Heel (EH)

Possible HFCSbetween 0 and 3

Mid Foot Contracture (MFC)

1.Curved lateral border

2.Medial crease

3.Coverage of the talar head

Possible MFCS between 0 and 3

Bracing Protocol

Babies were then shifted to Maintenance phase 3 weeks after tenotomy by bracing them in dennisebrownesplint ; The splint is to be used 23 hours a day for the first 3 months and then atleast14 hours a day for 3 years.

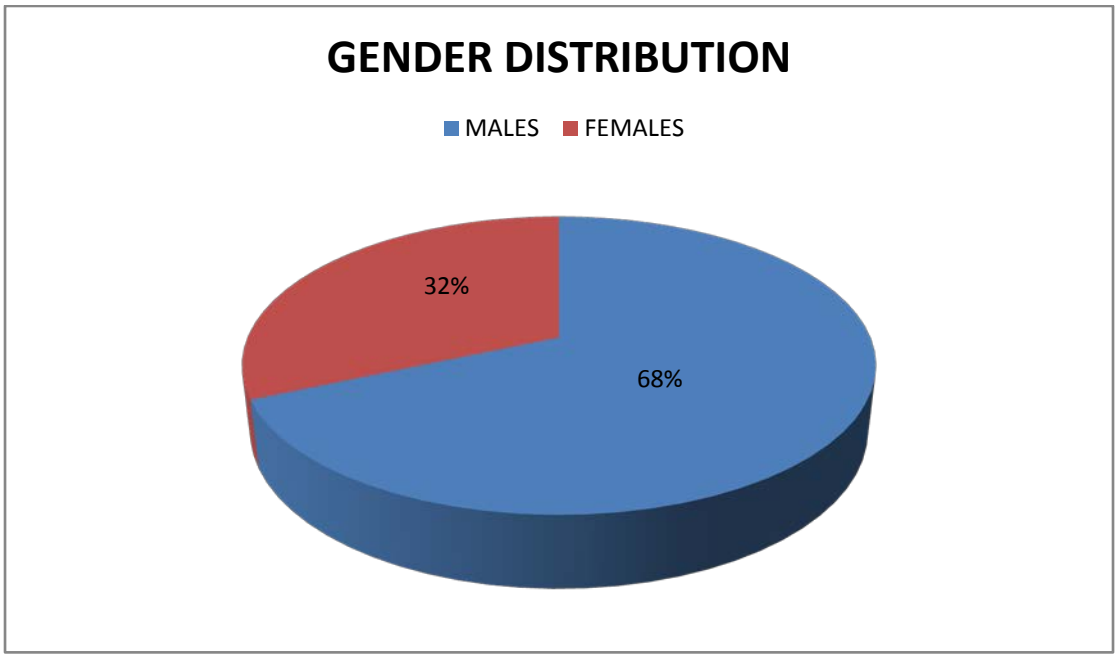
RESULTS

All 38 patients were managed by serial cast correction by ponseti technique using the Pirani scoring for assessing the results. The following were the observations made during the study.

GENDER DISTRIBUTION:

Male	Female
26	12

Table - 1

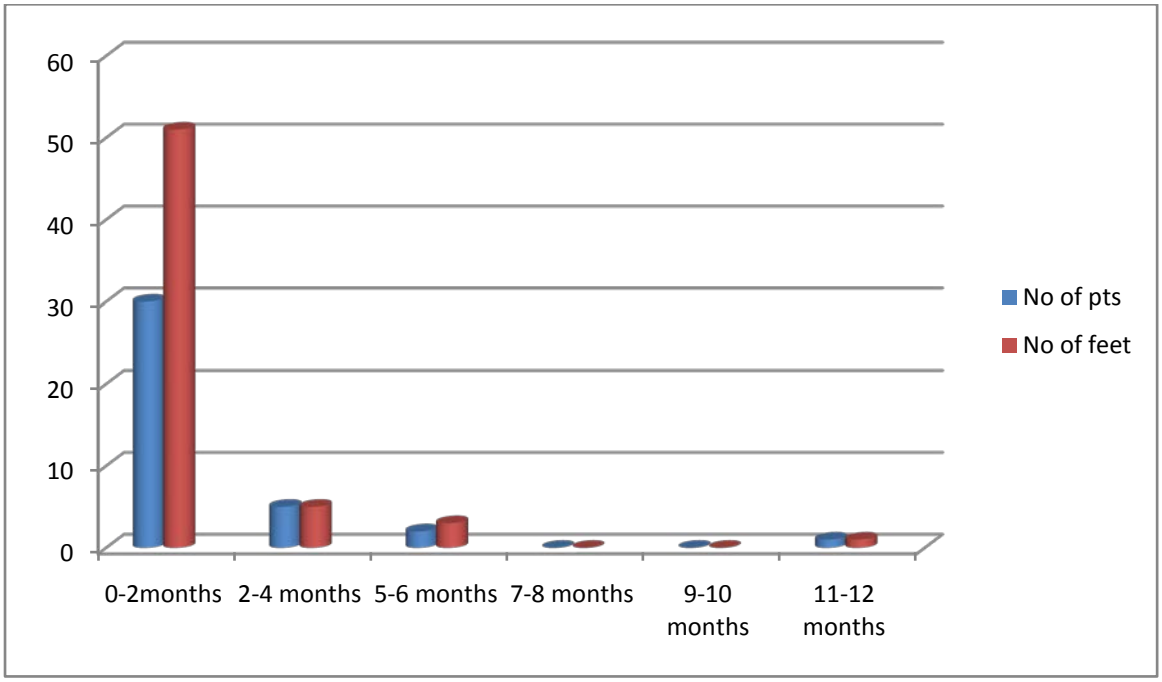


AGE DISTRIBUTION:

Age at Presentation (In months)	No. Of Patients	No. Of Feets
0-2 months	30	51
3-4 months	05	05
5-6 months	02	03
7-8 months	0	0
9-10 months	0	0
11-12 months	01	01

Table - 2

Of the children who presented to us, 79% (30 out of 38 babies) were below 2 monthsof age and 30% above 2 months

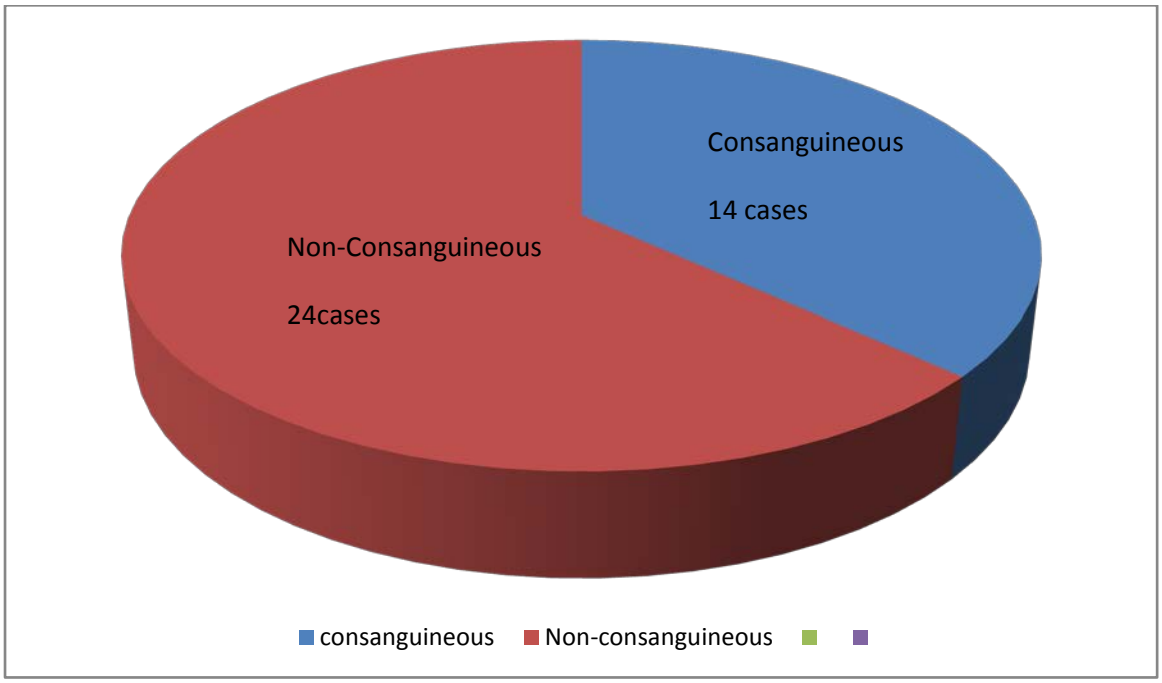


The age at presentation has significance with respect to outcome. Those children who presented less than 2 months had good results manifested by the difference between initial and final Pirani.

CONSANGUINITY:-

Consanguineous	Non-consanguineous
14	24

Table - 3

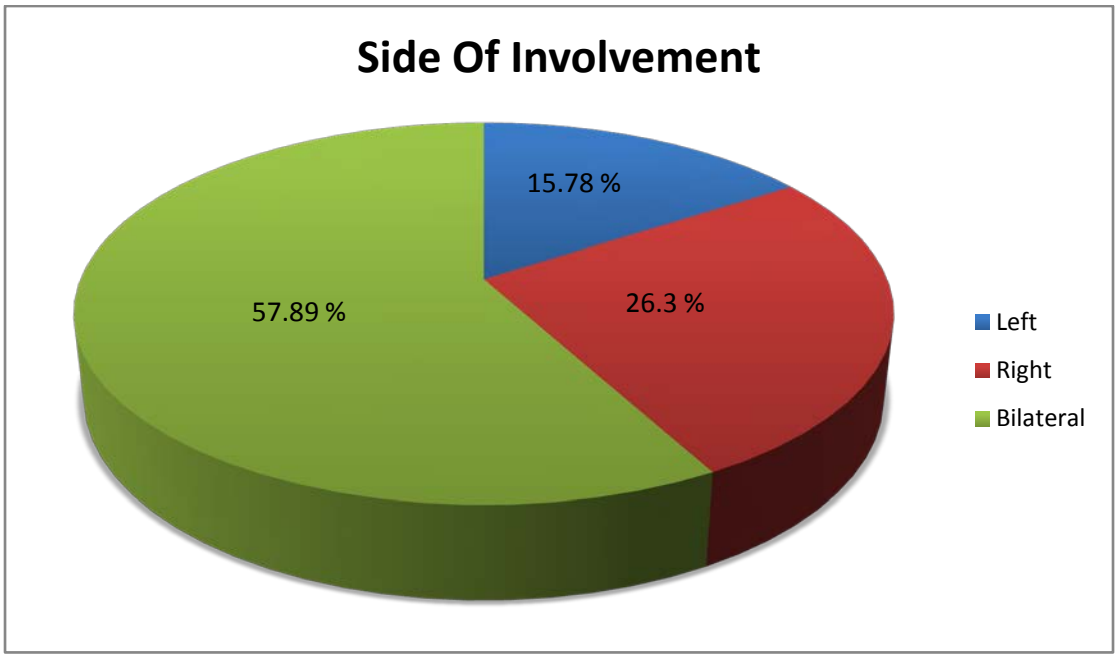


Of the 38 cases only thirty seven percent were born out of consanguineous marriage.

SIDE OF INVOLVEMENT:

Side	Left	Right	B/L
No. of cases	6	10	22

Table - 4



In our study predomininant bilaterality was seen in 57.89% of cases .

15.78% were left sided and 26.3% were right sided. The Ponseti Method for the Management of CTEV – 10 year Results Presented in National Medical Students Paediatric Conference (NMSPC) 2014, Brighton, UK reports a 50% bilaterality

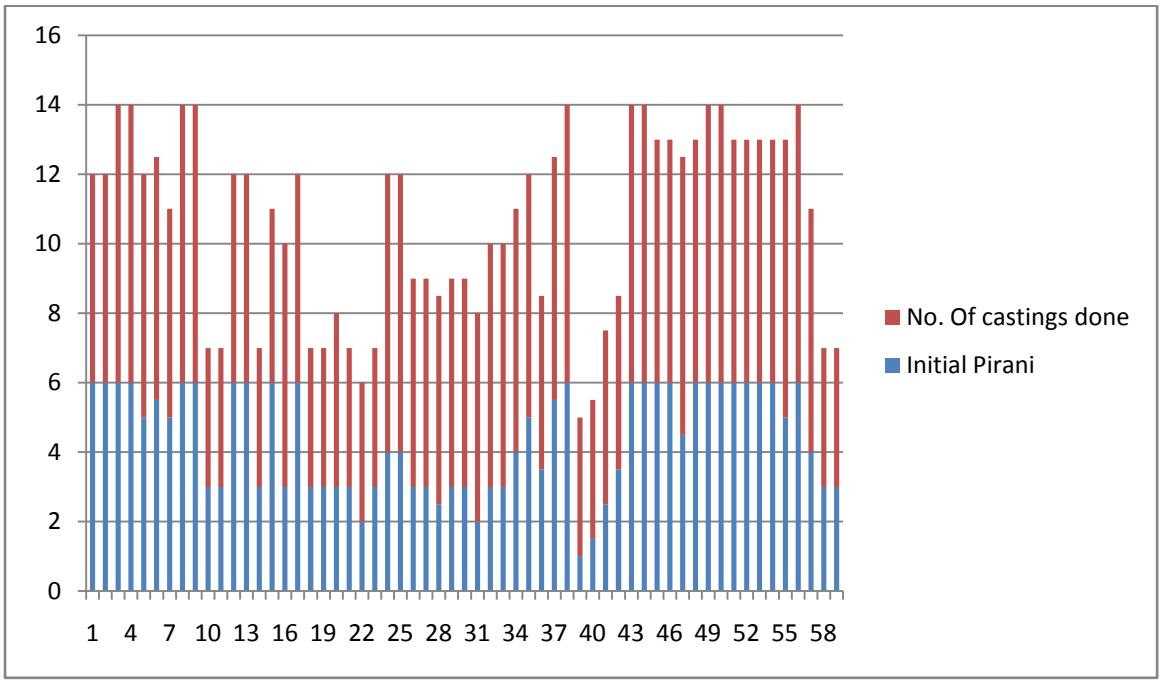
Relationship between Age at presentation and Final result:

Age at Presentation (In months)	Mean initial Pirani	Mean final Pirani score
0-2 months	4.098	0.088
3-4 months	3.6	0.40
5-6 months	12	0.25
7-8 months	-	-
9-10 months	-	-
11-12 months	06	0.5

Table - 5

The younger age (<2mon) group fared better in terms of results on comparing the mean initial pirani score and the mean final pirani score.

5.Initial Pirani and No. of Castings needed:

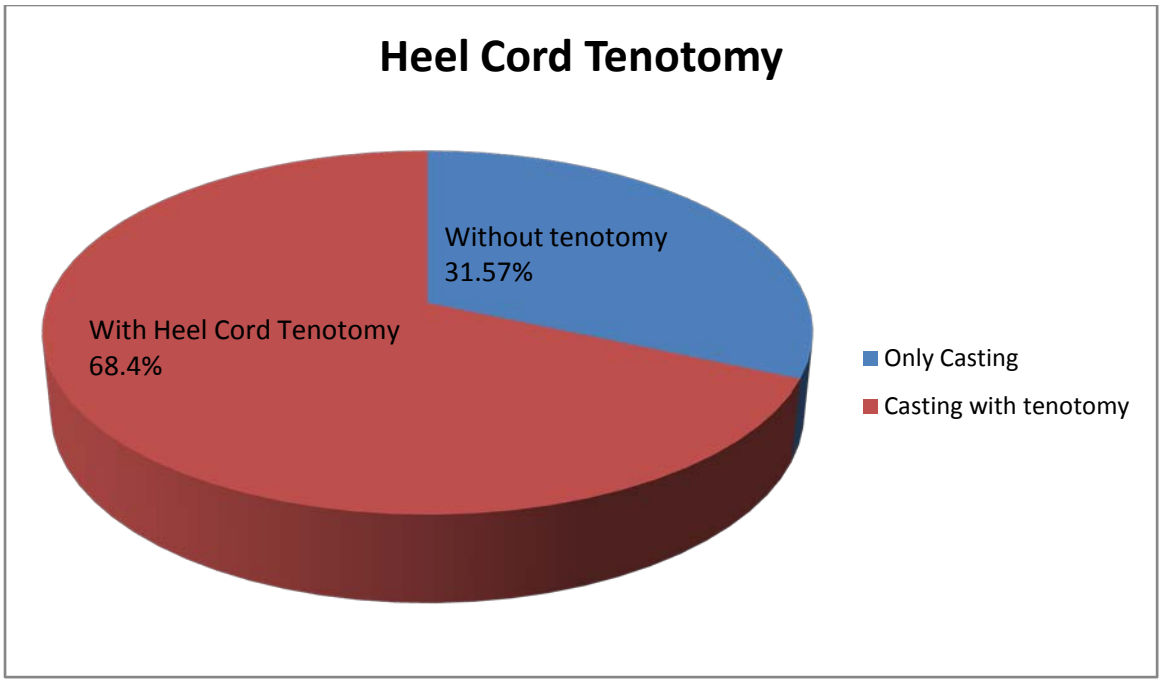


It is observed that the lesser the Pirani score at presentation – the lesser will be the number of castings needed for correction. The average Number of casts per foot was 6.15.

Percutaneous Tenotomy:

Treatment	No of cases	Percentage
Only Casting	12	31.57%
Casting &Tenotomy	26	68.4%

Table - 6



In our study we were able to achieve correction in 31.57% of the cases without resorting to heel cord tenotomy.

Associated Conditions :

Conditions	No of patients
Cleft Lip	2
DDH (B/L)	1
Omphalocele	1

Table - 7

In our study the most common associated finding was cleft lip seen in two of our cases.

Complications:

Complication	No of patients
Superficial sores	3
Crowding of toes	2

Table - 8

Minor complications were noted in 13.15% of our cases. The superficial sores were managed with further castings with adequate soft padding and allowing the skin to heal. The crowding of toes was managed but allowing enough space for the toes especially the dorsum for free toe movements.

CASE – I

Name	:	B/O Sangeetha
Age at presentation	:	7/365 days
Sex	:	Female child
Laterality	:	Bilateral
Consanguinity	:	Yes
Tenotomy	:	Done
Bracing	:	Applied

CASE – I

AT PRESENTATION



THIRD VISIT


TENOTOMY FOLLOWED BY BRACE APPLICATION


AT 1 YEAR FOLLOW-UP

CASE – II

Name	:	B/O Thangaponnu
Age at presentation	:	19/365 days
Sex	:	Male child
Laterality	:	Bilateral
Consanguinity	:	Yes
Tenotomy	:	Done
Bracing	:	Applied

CASE – II

AT PRESENTATION


SECOND VISIT



TENOTOMY FOLLOWED BY BRACE APPLICATION



AT 5 MONTHS FOLLOW-UP


CASE – III

Name	:	B/O nithya
Age at presentation	:	11/365 days
Sex	:	Male child
Laterality	:	Bilateral
Consanguinity	:	No
Tenotomy	:	Done
Bracing	:	Applied

CASE – III

AT PRESENTATION


SECOND VISIT


TENOTOMY


AT 11 MONTHS FOLLOW-UP


CASE – IV

Name	:	B/O Veerammal
Age at presentation	:	5 months
Sex	:	Male child
Laterality	:	Bilateral
Consanguinity	:	No
Tenotomy	:	Not done
Bracing	:	Applied

CASE – IV

AT PRESENTATION

THIRD VISIT

FOURTH VISIT

AT 7 MONTHS FOLLOW-UP

CASE – V

Name	:	B/O Shakthi priya
Age at presentation	:	03/365 days
Sex	:	Male child
Laterality	:	Right Sided
Consanguinity	:	No
Tenotomy	:	Not Done
Bracing	:	Applied

CASE – V

AT PRESENTATION


SECOND VISIT


THIRD VIST


AT 7 MONTHS FOLLOW-UP

CASE – VI

Name	:	B/O Meena
Age at presentation	:	1 month
Sex	:	Female child
Laterality	:	Bilateral
Consanguinity	:	Yes
Tenotomy	:	Done
Bracing	:	Applied

CASE – VI

AP PRESENTATION

THIRD VISIT

FOURTH VISIT FOLLOWED BY TENOTOMY

POST TENOTOMY APPEARANCE AND AT 8 MONTH FOLLOWUP

CASE – VII

Name	:	B/O Anjali
Age at presentation	:	1 month
Sex	:	Female child
Laterality	:	Bilateral
Consanguinity	:	No
Tenotomy	:	Done
Bracing	:	Applied

CASE – VII

AT PRESENTATION

THIRD VISIT

TENOTOMY

AT 8 MONTH FOLLOW-UP

DISCUSSION

The method of serial cast correction for Club foot was introduced and developed by by Ignacio Ponseti, MD, at the University of Iowa in 1950s. It was applied to infants with congenital clubfoot deformity with the aim of achieving a plantigrade, functional foot by non surgical methods Satisfactory functional outcomes have been reported in 85-90% of the cases undergoing serial cast correction as per schedule..Nonsurgical treatment regimens of manipulation and casting at other institutions have had less success and have been associated with such complications as increased cavus deformity, false correction with mid tarsal breach and rocker-bottom deformity, flattening of the talar dome, pressure sores from casts, and even fractures secondary to excessive force during manipulation

Since the early 1970s, this has led to a trend toward surgical intervention in cases of congenital clubfoot, primarily in the form of the posterior and medial soft-tissue releases as described by Turco with modifications by Crawford et al and McKay and Surgery is usually performed at 6 months to 1 year of age. Extensive surgical release carries both immediate and long-term inherent risks. Wound complications including scarring, infection, neurovascular compromise, and avascular

necrosis may occur in the immediate postoperative setting and Overcorrection with calcaneus deformity, heel valgus, pes planus, and forefoot abduction, and under correction with persistent equinus, heel varus, and metatarsus adductus may also occur and . Because these complications have become more readily evident, a renewed interest in nonsurgical treatment of congenital clubfoot has occurred.

In our series, we have treated 38 babies with idiopathic clubfoot by ponseti method by serial casting. Of the 38 babies 22 had bilateral affection and 16 had unilateral. 26 of the babies were male and 12 were female.

Out of the 38 babies, 30 presented within first 2 months with 51feets, 5 babies presented between 3rdand 4thmonth with 5feets and 2 of them presented later at 5-6 weeks with 3 feet. One Unilateral CTEV patient presented late around 10 months of age.

Ponseti has reported a relapse rate of 78% in patients noncompliant with the straight-last shoe and abduction bar regimen and a relapse rate of 7% in compliant patients. All of the noncompliant patients in Ponseti's series were corrected with recasting. We had recurrence of fore foot adduction in 6 of our patients(15.7%) probably reflecting a better compliance with brace. Porsche etal[80] described a relapse rate of 28% in his study. The relapsed foots required additional castings but finally all the feet were supple and fitted properly within the Dennise Browne splint. .

Study group		**Relapse rate (%)**
Ponsetti	Brace compliant	7
	Brace Non-compliant	78
Porsche etal		28
Our study		15.7

Table - 9

GENDER DISTRIBUTION:

In our series the male to female ratio is not very high (Male:female = 2.2:1).in comparison to the series of Yamamoto [41] (male:female, 3:1),Chesney D et al42(2:1). Palmer[43] found the sex correlation to be insignificant. Ignorance, social bias and increased. attention towards males in our region can account for the higher incidence in males in Indian setup.

Study group	**Males**	**Females**
Yamamoto etal	3 : 1	
Chesney D etal	2:1	
Palmer etal	Insignificant	
Our study	2.2:1	

Table - 10

The number of cast per feet in our study was four to seven (average 6.15 casts per foot). In another study by Laaveget al.45 the mean number of casts during their treatment was seven.Morcuende46,47 reported that 90% of the patients required five or fewer casts.. Over a period of time, as part of the learning curve people have started changing plaster casts at shorter intervals and fewer casts per feet give faster results. Those feet which required a greater number of casts in our study had a high Pirani score at the onset of treatment. Also we found correlation between late presentation and the higher number of casts. The duration initially was high which decreased over time reflecting a steady learning and started getting faster correction.

Of the children who presented to us, 79% (30 out of 38 babies) were below 2 monthsof age and 30% above suggesting a probably deficient referral system in our area and ignorance on the partof the parents.

Tenotomy:

Tenotomy was required in 68.4% of the cases (26 out of 38 feet). Pirani carried out tenotomy in over 90% of his clubfoot patients.

Laaveg et al.did tenotomy in 78% cases. In the study by Dobbs et al.tenotomy was required in91% cases;

Relapse / Reccurence:

Of the 38 cases 6 feet had recurrence of forefoot adduction, which required additional castings but finally all the feet were supple and fitted properly within the Dennise Browne splint. .

3 babies had developed pressure sores because of cast which healed uneventfully. Repeat correction and casting was done after 2 weeks for them.Wallace.B.Lehman in his study on club foot puts the incidence of compilcations to be 10.2%. Alexis bandore shsville etal in their study gave an complication rate of 50%.

CONCLUSION

The ponseti method of serial cast correction for CTEV is an excellent method as per our study. In a developing country like India, the method is very safe, economical, easy and result oriented method.

- The earlier the child presents the quicker will be the correction and better will be the result.
- The less severe types with low pirani scores achieves a quicker correction with less number of casts.
- Correction initiated by ponseti technique at an earlier age and adhered to regular weekly casting protocol tends to give better functional and cosmetic results.
- Even relapses can be managed with further castings alone.

"Thus we conclude that the Ponseti method is a very safe, efficient and economical treatment for the correction of club foot that radically decreases the need for extensive corrective surgeries. The Ponseti method of cast correction is important especially in developing countries as it is effective and inexpensive.The results are excellent when treatment is begun early"

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Proforma - Case Sheet

MANAGAMENT OF IDIOPATHIC CLUBFOOT BY PONSETI METHOD

Name:	Age at presentation:
Father's name:	Sex:
Mother's name:	Address:
Informed Consent :	Socio-economic status:
Date of presentation :	

---

(1) Family History :	Consanguinity	Y/N
	Birth Order	
	Affected Parents	Y/N
	If yes, Mother / Father /Both	
	Affected Siblings	Y/N
(2) Antenatal History	Uneventful	Y/N
If not,	Exposure to Drugs	Y/N
	Polyhydramnias	Y/N
	Oligohydramnias	Y/N
	Twin Pregnancy	Y/N
(3) Labour Details	Presentation Breech / Cephalic	
	FTND / LSCS	

(4) Systemic Examination

RS

CVS

P/A

Hips

Spine

Other Deformities

Associated Neurological abnormalities

(5) Local Examination

Unilateral / Bilateral

If Unilateral, Affected Side: Right / Left

Pirani Scoring on Presentation Right Left

Midfoot Score :

Medial Crease

Lateral Curvature

Talar Head Coverage

Hindfoot Score

Empty Heal

Rigid Equinus

Posterior Crease

(7) No. of Visits (Weekly)

1 2 3 4 5 6

Pirani Score and Correction achieved at every visit

(8) TA Tenotomy Done Y/N

Anaesthesia Local / Sedation

Immobilisation for 3 weeks

(9) Pirani Scoring Post Correction

Midfoot Score :

Medial Crease

Lateral Curvature

Talar Head Coverage

Hindfoot Score

Empty Heal

Rigid Equinus

Posterior Crease

(10) Denise Browne Splint compliance

First 3 months	24 hrs	Y/N
After 3 months	Nightly application	Y/N

(11) a. Monthly Visits

1 Correction Maintained / Relapsed

2 Correction Maintained / Relapsed

b. Tri - monthly visits

1 Correction Maintained / Relapsed

2 Correction Maintained / Relapsed

(12) Remarks :

SL N0	IP.No.	NAME	AGE	SEX	BIRTH ORDER	CONNSANGUINITY	SIDE	ASSOCIATIONS	PREVIOUS TREATMENT	PIRANI SCORE (R/L)	TENOTOMY	NO OF CASTS	FINAL PIRANI	FOLLOW-UP
1	28897	B/O Sangeetha	7/365	F	1	Yes	B/L	Nil	Nil	3/ 2.5	Yes	6	0/0	14 mon
2	17778	Baby Logesh	3/365	M	1	yes	B/L	Nil	Nil	3/3	Yes	6	0/0	14 mon
3	4237	B/O Janani	4 Mon	F	1	yes	L	Nil	Ponsetti	2	No	6	1	14 mon
4	5429	B/O Veerammal	5 mon	M	2	No	B/L	Nil	Nil	3/3	No	7	0.5	7 mon
5	581	B/O Nithya	11/365	M	2	No	B/L	Nil	Nil	4/5	Yes	7	0	11 mon
6	0718	B/O Kasthuri	5/365	M	2	No	Rt	Nil	Nil	3.5	Yes	5	0	10 mon
7	83903	B/O Sakunthaladevi	4/365	M	1	Yes	Rt	Nil	Nil	5.5	No	7	0	10 mon
8.	65516	B/O Chinnaponnu	3 mon	M	1	No	Rt	Nil	Nil	6	No	8	0.5	9 mon
9	472814	B/O Meena	1 mon	F	1	Yes	B/L	Nil	Nil	1/1.5	No	4	0	8 mon
10	6756	B/O Deepa	5mon	M	1	No	B/L	Cleft lip	No	2.5/3.5	Yes	5	0	8 Mon
11	33162	B/O Bhuvaneswari	10/365	M	1	No	B/L	Omphalocele	Nil	6/6	Yes	8	1	7 mon
12	09982	B/O Palaniyammal	2/365	F	2	Yes	B/L	Nil	No	6/6	Yes	7	0	8 mon
13	5210	B/O Shakthipriya	3/365	M	1	No	Rt	Nil	Nil	4.5	No	8	0	7 mon
14	2771	B/O Jegathambal	3/365	M	1	No	Lt	Nil	Nil	6	No	7	0	6 mon
15	72726	B/O Thangaponnu	19/365	M	2	Yes	B/L	Nil	Nil	6/6	Yes	8	0/0	5 mon
16	332793	B/O Anandhi	15/365	F	1	No	B/L	Nil	Nil	6/6	Yes	7	0.5	6 mon
17	72553	B/O Anjali	1 mon	F	2	Yes	B/L	Nil	Nil	6/6	Yes	7	0/0	6mon 2 weeks
18	23345	BO Vijayalakshmi	2 mon	F	2	No	Lt	Nil	Nil	5	No	8	0	5mon
19	45584	B/O Sakunthala	10 mon	M	2	Yes	Rt	Nil	Nil	6	No	8	0.5	5 mon
20	6479	B/O Vasugi	2 mon	M	1	No	Lt	B/L DDH	Nil	4	No	7	0.5	5 mon

SL N0	IP.No.	NAME	AGE	SEX	BIRTH ORDER	CONSANGUINITY	SIDE	ASSOCIATIONS	PREVIOUS TREATMENT	PIRANI SCORE (R/L)	TENOTOMY	NO OF CASTS	RESULTS	FOLLOW-UP
21	27797	B/O Kalaiarasi	3/365	M	1	No	B/L	Nil	Nil	6/6	Yes	6	0/0	5 mon
22	15954	BabyHariharan	2 mon	M	1	No	B/L	Nil	Nil	6/6	No	8	0.5/0	5 mon
23	71622	B/O Deepa Kumar	3/365	F	1	No	B/L	Nil	Nil	5/5.5	Yes	7	0/0	5 mon
24	72333	Baby Kanishka	3 mon	F	1	No	Rt	Nil	Nil	5	Yes	6	0.5	4 mon 2 weeks
25	28714	B/O Umarani	6/365	M	1	No	B/L	Nil	Nil	6/6	Yes	8	0/0	4 mon 2 weeks
26	29536	B/O Vasantha Rani	I mon	F	1	yes	B/L	Nil	Nil	3/3	No	4	0/0	4 mon
27	32500	B/O Meena	1 mon	F	1	No	B/L	Nil	Nil	6/6	Yes	6	0.5/0.5	6 mon 2 weeks
28.	3700	B/O Parvathi	3/365	M	3	No	B/L	Cleft lip	Nil	3	Yes	4	0/0	6 mon
29	58891	Baby Vijay	22/365	M	1	No	Rt	Nil	Nil	6	Yes	5	0	6 mon 2 weeks
30	62931	BO sasikala	2 mon	M	1	No	Lt	Nil	NIl	3	Yes	7	0.5	6 mon
31	60276	BO Priya	1 mon	M	1	yes	Rt	No	Nil	6	Yes	6	0	6 mon
32	246805	BO Jeyalakshmi	3/365	F	1	No	B/L	No	Nil	3/3	Yes	4	0/0	6 mon
33	33696	BO Harini	10/365	M	1	No	Rt	No	Nil	3	Yes	5	0	6 mon
34	35993	BO Kannagi	4 mon	M	2	Yes	Rt	No	Nil	3	Yes	4	0	8 mon
35	36555	BO Kavitha	3 mon	M	2	Yes	Lt	No	Nil	2	Yes	4	0	8 mon
36	38123	BO Panchali	8/365	M	1	Yes	B/L	No	Nil	3	Yes	4	0/0	8 mon
37	39000	BO Bhuvana	10/365	M	2	No	B/L	NO	Nil	4/4	Yes	**8**	**0.5/0**	8 mon
38	42002	BO Mary	11/365	M	2	No	B/L	No	Nil	3	Yes	**6**	**0/0**	8 mon

B/L – BILATERAL, B/O Baby Of